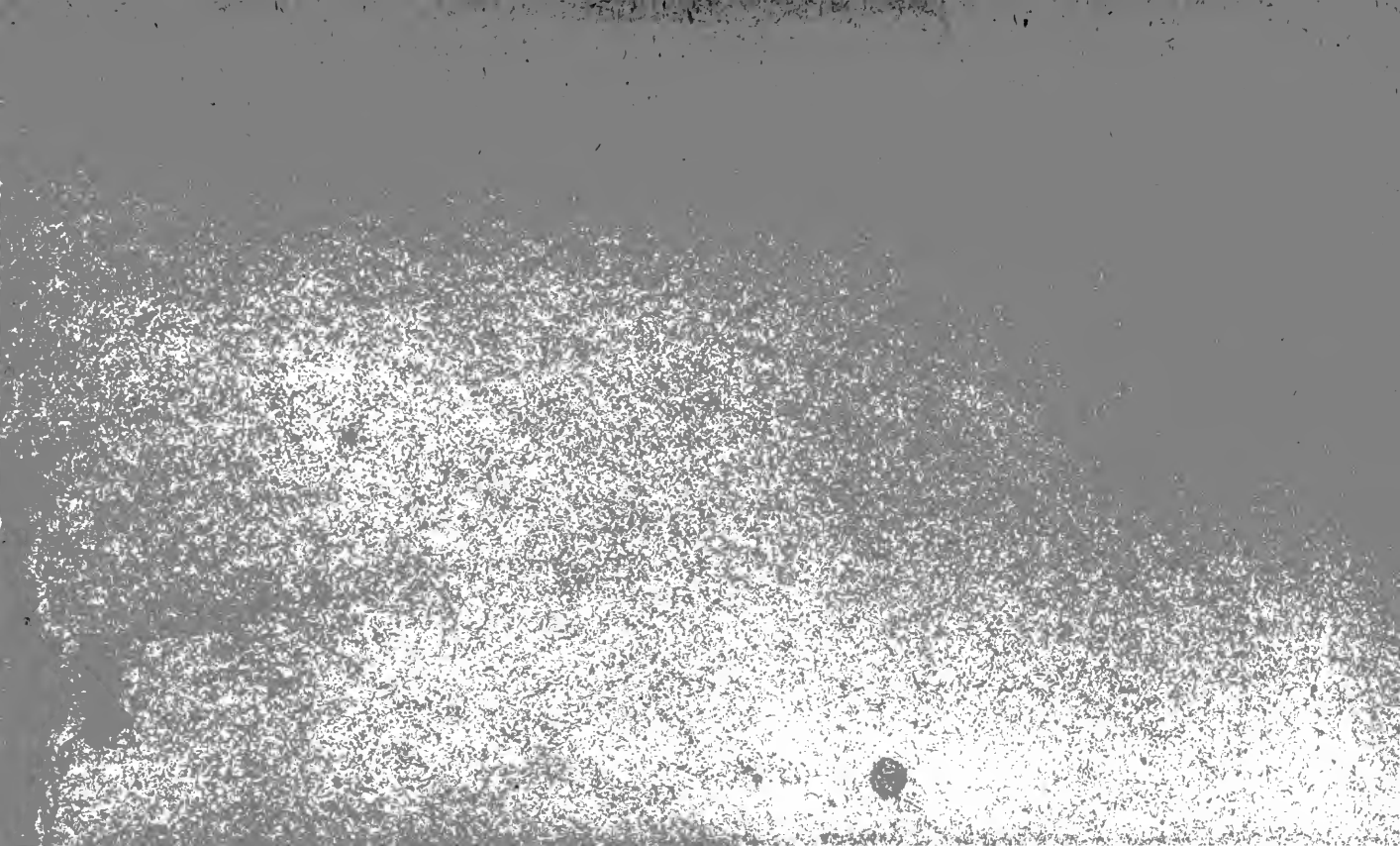
GLAUCOMA

A HANDBOOK FOR THE PRACTITIONER

R. H. ELLIOT

Isaac McCann



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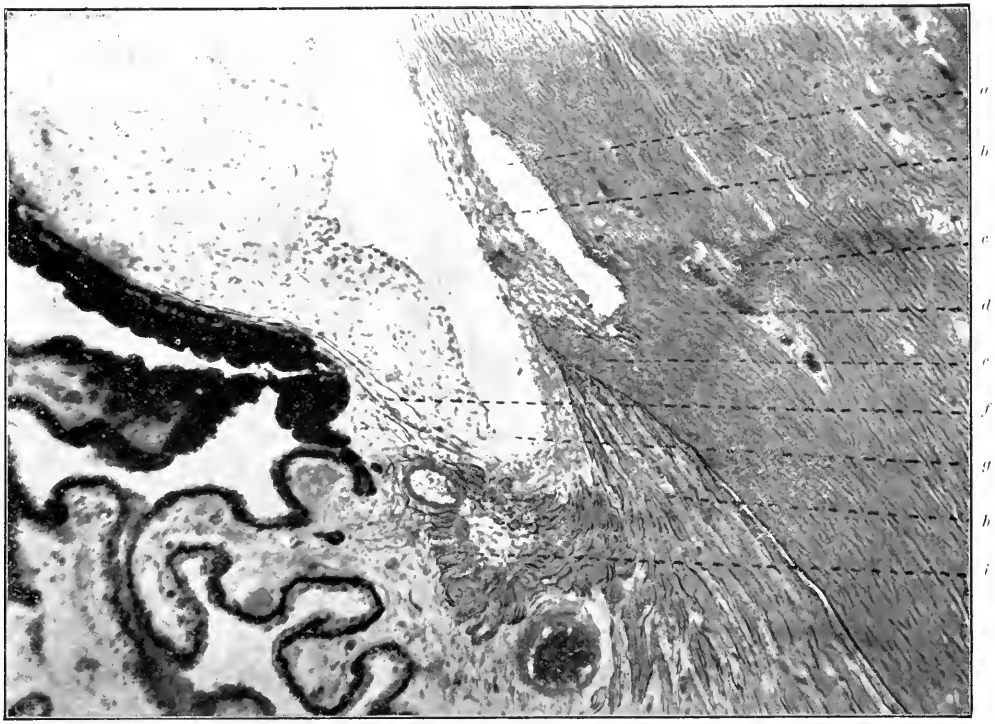

FIG. 1.—THE FILTRATION ANGLE.

a, Canal of Schlemm; *b*, trabecular tissue of pectinate ligament; *c*, scleral vein; *d*, dense scleral tissue; *e*, scleral process or spur projecting inwards and forwards from the sclera close behind the canal of Schlemm; *f*, radial muscular fibres of the iris extending outwards below the iridial angle; *g*, iridial angle; *h*, meridional fibres of the ciliary muscle; *i*, circular fibres of the ciliary muscle.

(By kind permission of Professor Arthur Thomson, M.A., M.B., F.R.C.S., and the Oxford Press.)

GLAUCOMA

A HANDBOOK FOR
THE GENERAL PRACTITIONER

BY

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To

E. C. I. E.

To

SYDNEY STEPHENSON, M.B., C.M., F.R.C.S., D.O.

THIS

BOOK IS DEDICATED

IN GRATEFUL ACKNOWLEDGMENT OF THE VERY GREAT SERVICES WHICH HE HAS RENDERED TO OPHTHALMOLOGY IN GENERAL, AND TO THE STUDY OF GLAUCOMA IN PARTICULAR, IN THE PAGES OF "THE OPHTHALMOSCOPE," DURING THE FOURTEEN YEARS OF THE VERY SUCCESSFUL APPEARANCE OF THAT JOURNAL

PREFACE

During the last ten years a very extraordinary amount of interest has been taken in the subject of glaucoma. At no time in the history of ophthalmology has this been equalled, save possibly in the ten years which followed the declaration of von Graefe's great discovery. Between these two periods of interest in glaucoma, dating from the start of each, nearly half a century elapsed. Though this period was marked by no spectacular event in connection with this particular subject, it was none the less a time of steady progress and of fruitful speculation. Some of the questions which puzzled von Graefe can be answered by a student to-day, and yet we are far from having attained finality. There are still many matters on which authorities are at variance. There are, fortunately, many more on which they are in agreement.

It is not the purpose of such a work as this to discuss the former. Space and common sense alike forbid such a course. The aim which I have kept before me has been to be of service to *the busy medical practitioner*. The importance of the subject appears to me to demand the publication of a monograph. My object has been to write the book simply and concisely, so that all may understand it, shortly, so that all may have time to read it, and without bias, so that all may feel its appeal.

Discussions of disputed points have been avoided; the views expressed are those which are current amongst the great majority of ophthalmologists. Names of

authorities have been purposely omitted. A certain amount of dogmatism has been inevitable.

The material for a much more exhaustive treatise on the same subject lies on my desk to-day. The appeal of that work will be to the scientific ophthalmologist. In it subjects will be discussed at length, and authorities freely quoted. Its aim will be widely different from that of this little book, but the world-conflict makes the publication of such a work an impossibility at present. It was to have been issued first, and this book was to have followed it. Circumstances have been too strong for the carrying out of this intention; and now that the data for the larger work are all ready to hand, there is nothing to hinder the publication of the small one, which, after all, is but an epitome of the former. The book is addressed to the busy general practitioner in the hope (1) that it will put him on his guard, so that he may not overlook cases of glaucoma when he meets them; (2) that it will teach him to make sure of his diagnosis in case of doubt; and (3) that it will serve to indicate to him the latest lines along which the treatment of the disease is conducted to-day.

54, Welbeck Street,
Cavendish Square, W.,
January, 1917.

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INTRODUCTORY

THE term "glaucoma" is not the title of any one single disease. It is rather a convenient clinical label for a large group of pathologic conditions, the distinctive feature common to all of which is a rise in the intra-ocular pressure.

The causes of these conditions are many and varied, the pathological findings are most diverse, and the difference in the symptoms presented is so extraordinary, that very careful study is required to detect the bond which serves to unite these very dissimilar manifestations of disease in a common category.

When we speak of the hardness of a glaucomatous eye, or of its rise in tension, we are referring to the outward manifestations of an increase of the fluid pressure within the globe. To this increase all the causes of glaucoma lead up; on it every sign and symptom of the condition depend.

If the rise in pressure can be traced to the action of some antecedent local disease, we speak of the glaucomatous condition as **secondary**; failing this we term it **primary.**

The presence of an increase in intra-ocular pressure necessarily brings about some measure of interference with the free escape of blood from the interior of the eye

to the surface. So long, however, as such interference does not give rise to obvious congestion of the eye or of its conjunctiva, we speak of the condition as **"simple** or non-congestive glaucoma." When evidence of interference with the venous return makes its appearance, the disease is said to be **congestive.** The term "inflammatory," though often used in this connection, is erroneous and should be dropped.

The classification of all cases of glaucoma into three groups—viz., the **acute,** the **subacute,** and the **chronic**—has been productive of much confusion, owing to the difference in the way in which these terms have been used by writers on the subject.

Any case which presents the signs and symptoms of severe congestion, as a result of a steep rise in intraocular pressure, may reasonably be spoken of as acute; similarly with eyes presenting the signs of subacute congestion. To define a chronic case is more difficult. All that the term really implies is that the condition has lasted for some time; but, in the accepted use of the term, it is also understood that the case is not in an acute or subacute stage. Any glaucomatous eyeball, whether of the simple or of the congestive type, may fall into this category. The point to be emphasized is that there is no such thing as acute, subacute, and chronic glaucoma. Any glaucomatous eye may be in an acute, a subacute, or a chronic stage, and may readily pass from one to another of these stages, and back again into that from which it sprang; but to speak of acute, subacute, and chronic glaucoma, as if we were dealing with so many clinical entities, is wrong and misleading.

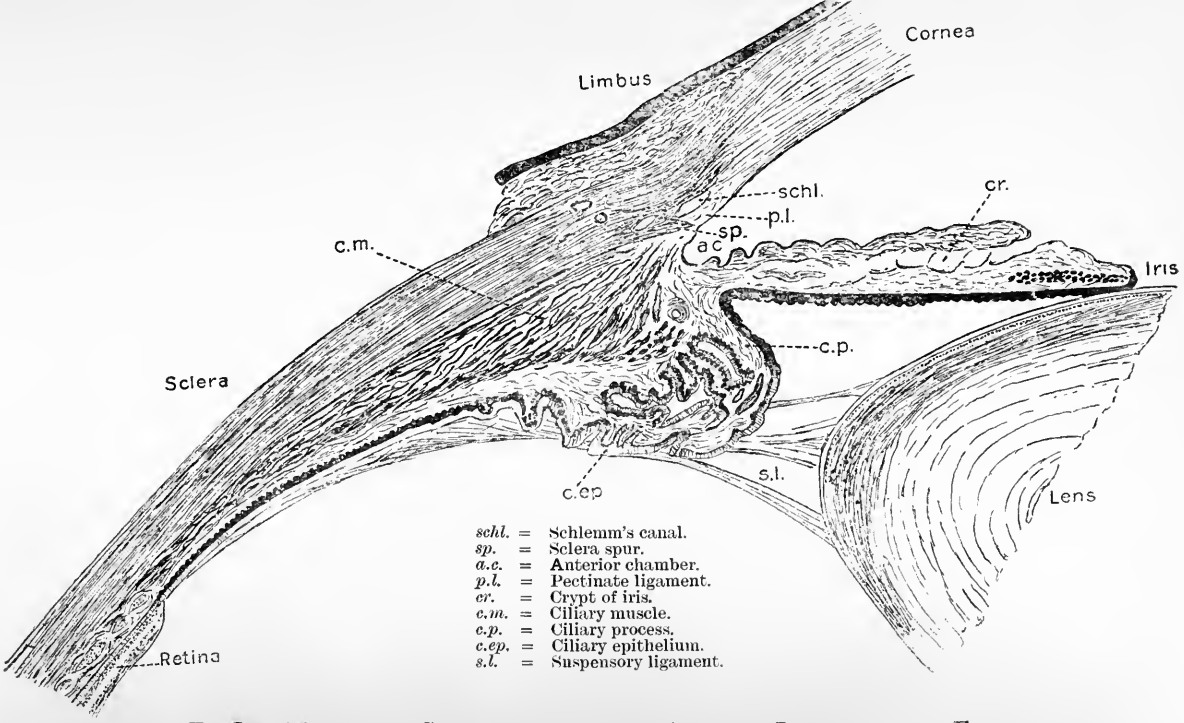


FIG. 2.—MERIDIONAL SECTION THROUGH THE ANTERIOR PORTION OF THE EYE.

(*Modified from Fuchs by E. E.*)

[*To face p.* 3.

CHAPTER II

THE ANATOMY OF THE PARTS CONCERNED IN GLAUCOMA

BEFORE we can study the processes of disease in an organ, or the methods of combating them, it is essential that we should make ourselves acquainted with certain anatomical details of the structures concerned.

The ciliary body and the parts adjoining it are "the cockpit" of glaucoma. A study of the two illustrations given will make a number of important points clear; a few of the more essential anatomical features demand some notice.

The Conjunctiva.—It will be noted in the illustration (Fig. 2) that the subconjunctival tissue is continued well in advance of the sclero-corneal junction. It is along this layer that we work in "splitting the cornea" in the operation of trephining, and the looseness of the tissue just referred to explains the ease with which that manœuvre is conducted.

Schlemm's Canal (Figs. 1 and 2) lies at the junction of the cornea and sclera, close to the inner surface of the corneo-scleral envelope. It is separated from the aqueous chamber only by the loose open network of the pectinate ligament (Figs. 1 and 2). Fluid can pass readily from the chamber to the canal through the open spaces of this meshwork.

The Scleral Spur (Fig. 1) lies between the pectinate ligament anteriorly and some fibres of the ciliary muscle

posteriorly. A contraction of these fibres will pull back the spur and tend to open wide the canal of Schlemm. As soon as muscular contraction ceases, the pectinate ligament being elastic, will draw the scleral spur back into place, and so close the canal of Schlemm (Arthur Thomson). It is suggested that the pump action above described is repeatedly in action during life, drawing the fluid from the anterior chamber into the canal of Schlemm, and then sweeping it on again from the canal into the neighbouring veins.

The Angle of the Chamber (Fig. 2) is at the best of times a narrow space. Inasmuch as the outflow of the aqueous fluid takes place in this neighbourhood, it is most important that the angle should remain widely open. Its patency may be infringed in several ways.—

1. *Extreme dilatation of the iris*, by crowding the membrane out to its periphery, tends to fill up the angle, and so to impede the passage of fluid through it.

2. The *ciliary body* may become swollen by congestion with blood. If it does so, its apices move forward. A glance at the illustration (Fig. 2) will show that, if this happens, these apices will press upon the base of the iris, and push the latter forward against the cornea, thus closing the angle. A second point may be learnt from the diagram. If the apices of the ciliary body move forward, the attachments of the suspensory ligament of the lens do the same; this, obviously, will allow the lens to advance, and so to press on the base of the iris.

3. As life advances, the *lens* enlarges, whilst the tunic of the eye remains stationary in size. The swollen lens tends to press the iris forward, and so to occlude the angle, just as in the previous case.

4. When the iris and cornea are brought into close apposition in one of the ways above described, their adjacent surfaces are apt to be glued together by the

exudate they throw out. In this way a permanent obliteration of the angle may be brought about.

The Iris.—On the anterior surface of this membrane are found pit-like depressions, the crypts of the iris (Fig. 2), which lead to the depth of the iris stroma, and which place its tissue spaces in free communication with the cavity of the aqueous chamber. In this stroma the fluid comes into intimate contact with the thin-walled iris veins, and passes into the latter by osmotic action. The crypts are of importance when the angle of the chamber is in an early stage of closure, for the fluid from the chamber can enter through them and find its way along the iris stroma to the neighbourhood of the pectinate ligament, across which it then passes to enter Schlemm's canal.

The Ciliary Body.—Whilst few authorities now accept the suggestion that there are definite tubular secretory glands in this body, it is believed that its lining cells have the power of taking up fluid transuded into their neighbourhood from the capillary vessels, and passing it across on to the free surface by a definite act of secretion.

The *fluid* thus poured out passes by two streams, one backward into the vitreous, and the other inward and forward into the posterior division of the aqueous chamber; thence it finds its way through the pupil into the anterior division of that chamber, and flows outward all round to reach the angle.

The ciliary body presents a large surface of attachment to the sclera. This is of interest in connection with the operation of *cyclo-dialysis*, the purpose of which is to tear the former structure away from the latter over the area marked out by a limited incision; the pectinate ligament is obviously divided during this step. The object aimed at is to open up a communication between the anterior chamber in front and the suprachoroidal

space behind, by means of the detachment of the ciliary body.

The *vessels and nerves* which supply the ciliary body and iris pass forward between the choroid and the sclera. In this course they are exposed to the full force of the intra-ocular pressure, since they lie against the hard unyielding scleral coat.

There is a free communication between the vascular system in the interior of the eye and that on its surface through the **perforating vessels,** which pierce the sclera close behind the cornea. Both here and in the neighbourhood of the vasa vorticosa, the evidence of congestion within the eye is to be looked for when present.

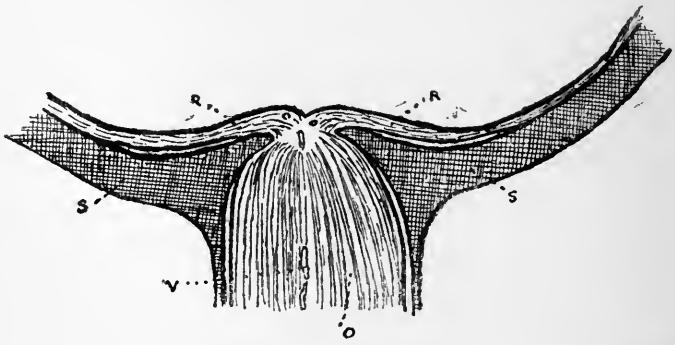


FIG. 3.—OPTIC NERVE ENTRANCE. (Drawn by E. E. from a lantern projection of a slide.)
R, Retina; *S*, Sclera; *O*, Optic nerve; *V*, Central vessel.

The whole of the **choroidal and retinal circulation** lies between the fluid contents of the eye and the unyielding sclera, and must suffer whenever the pressure within the eye is increased. The arterial and venous circulations react differently. The pressure diminishes the amount of blood entering the eye through the arteries, and also that leaving it through the veins. The effect is to diminish the arterial supply and to establish a condition

of venous congestion. If the pressure rises slowly, the circulation can adapt itself to the change of conditions, and the glaucoma remains simple. If, on the other hand, the increase in pressure is a rapid one, no such adaptation is possible, and an attack of congestive glaucoma is the result.

The **optic nerve entrance,** being the weakest part of the eye, is the first to yield under a rise of pressure, hence the cupping of the optic disc. The whole nerve-head, including the lamina cribrosa, is pushed backward Owing to the fact that the fibres of the optic nerve lose their medullary sheath as they pass through the scleral coat, the bulk of the nerve diminishes from without inward; consequently a section through this part shows the nerve presenting roughly the appearance of a truncated cone, with its base at the external surface, and its truncated end at the fundus (Fig. 3). The channel in which it lies has the same form. This explains the shape of an advanced glaucoma cup, the overhanging edges seen in the cup, and the apparent interruption in the continuity of the bloodvessels which emerge from it (*vide* Fig. 5, on p. 27, and Fig. 7B, on p. 29).

CHAPTER III

THE INTRA-OCULAR PRESSURE AND THE TENSION OF THE EYE

Before we can understand the ætiology or pathology of glaucoma, we must study the mechanism whereby the healthy eye is kept of its usual shape, and at a normal tension.

The tension of an eye is a measure of the state of distension of its tunic, and is a factor which can be estimated either by the fingers or by means of a special instrument, known as a tonometer.

The intra-ocular pressure, on the other hand, is the level of pressure at which fluid stands within the eye; this is constantly varying, and can only be measured by introducing the needle of a manometer into the globe, a procedure which for clinical purposes is manifestly out of the question.

Practically we measure the tension, and then seek to arrive at the intra-ocular pressure; but we must remember that the tension of a globe depends on other factors beside the pressure, the most important of these being the thickness and distensibility of the ocular tunic. We must therefore be very careful to keep the two terms distinct in our minds.

The Physical Conditions regulating Intra-ocular Pressure

The sclero-corneal coat of the eye constitutes a more or less unyielding tunic, which is, however, endowed with a degree of distensibility and of elasticity, properties

which are best marked in the young organ. The major part of the contents of the globe consists of the intra-ocular fluid. This fluid is constantly being changed; fresh quantities are always being poured out by the ciliary body, and a like amount is as constantly being drained away at the angle of the chamber, and from the surface of the iris. In this way a measure of equilibrium is kept up. Passing through this fluid, and freely bathed by it, are the bloodvessels of the eye.

It is probable that the intra-ocular fluid is poured out as the result of a **combination of filtration under pressure and active cell secretion.** As a result of the former, fluid is expressed from the capillaries of the ciliary body, and is accumulated in the neighbourhood of the epithelial cells lining the surface of that organ (*vide* Fig. 2); these in turn pass it through their bodies by a process of active secretion. The greater the difference between the capillary blood-pressure within the eye and the intra-ocular pressure, the more rapid will the transudation be. Moreover, a freer supply of blood to the ciliary body will excite its cells to a more active secretion.

The fluid finds its way back again into the circulation through the pectinate ligament, and the crypts of the iris (*vide* Fig. 1), which both lead into the veins of the eye. This return is effected partly by osmosis, and partly by a pump action, of which the motive power is supplied by the ciliary and iris muscles. These pull open the canal of Schlemm each time they contract, whilst the elastic rebound is furnished by the pectinate ligament and the scleral spur (Arthur Thomson).

There are thus two simultaneous and closely interdependent circulations going on within the eye—viz., (1) the vascular, and (2) that of the intra-ocular fluid. The latter is fed by, and returned to, the vessels of the former system.

The pressure of the intra-ocular fluid (known as "the intra-ocular pressure") depends on the volume of the total fluid (blood plus intra-ocular fluid) in the eye at any given time. If secretion is increased, or if excretion is impeded, and still more if both these factors are active, the volume of the intra-ocular contents increases, and can only then be accommodated by means of a slight but distinct distension of the external coat of the eye; to this distension there is opposed the elasticity of the ocular tunic. The pressure of the contents consequently rises, until the limit of distensibility and of elasticity is reached, when the eye becomes "stony hard."

Traced back to its first cause, the intra-ocular pressure depends on the blood-pressure within the eye. As in all other organs, this pressure is highest in the arteries as they enter the globe, and lowest at the points of exit of the veins therefrom. Speaking generally, the intra-ocular blood-pressure rises and falls with the general or systemic blood-pressure, but wide variations from this rule may occur.

It has already been pointed out that the difference in level between the capillary blood-pressure in the eye and the intra-ocular pressure (*i.e.*, the pressure of the free fluid within the eye) determines the freedom of secretion of the intra-ocular fluid. Therefore a rise in the capillary pressure will cause an increase in the volume of intra-ocular fluid, and *vice versa*, always provided that the intra-ocular pressure remains stationary. Conversely, a rise in the intra-ocular pressure will lower the rate of secretion, by tending to level up the capillary and intra-ocular pressures, and again *vice versa*.

It is essential to make it very clear that, though the systemic blood-pressure is of great importance, and is, indeed, the foundation element, it does not dominate

the whole situation. This will be better understood if we first consider certain side-influences.—

1. Any factor which obstructs the outflow of fluid from the angle of the chamber tends to increase the volume of the intra-ocular contents, and so to raise the pressure within the eye.

2. Any factor which facilitates the outflow from the angle of the chamber must have the opposite effect. In this connection it is important to remember that there is evidence that, when the intra-ocular pressure rises under physiological conditions, fresh channels of excretion open, or else the existing ones enlarge, and so the removal of the excess of fluid is facilitated, thereby relieving the pressure within the eye. It is highly probable that the same thing occurs under pathological conditions.

3. A rise in the intra-ocular pressure will tend to close the lumina of the veins at the points where their pressure is lowest—viz., just where they are passing out of the eye; for the pressure here is only a little above that of the surrounding intra-ocular fluid. We know this is so, because if we press lightly with a finger on the globe, whilst using an ophthalmoscope, we can see the lumina of the veins close up at their proximal ends. We infer that under ordinary conditions the pressure in the veins is kept at such a level that the current of blood can still just escape from the globe. The closure of the venous exits will result in a rise of the venous pressure, until the obstruction is overcome. Such a rise will work back through the capillaries towards the arteries. An obstruction to venous exit will therefore in the first instance bring about a rise in capillary blood-pressure. *Vice versa*, a fall in venous exit pressure will cause a fall in capillary pressure. Now, a rise in capillary blood-pressure favours an increase of secretion, provided other things are equal; whilst a fall in it cuts down secretion.

If these elements of the situation are once grasped, it is easy to understand how the intra-ocular pressure is normally maintained at a steady level, for each factor that would tend to raise or lower the pressure within the eye carries with it compensating influences. How, then, does it come about that the pressure of the eye ever rises far above the normal? A rise in intra-ocular pressure, as has been shown, closes the venous exits, and so raises the capillary blood-pressure. This should lead to an increased escape of fluid from the eye, but if, owing to the condition of the excretory channels, it cannot do so, the volumetric contents of the eye rise instead of fall, as they normally should. Once again the venous exits tend to become closed, and once again, in response, the venous blood is dammed back until the vascular pressure behind it suffices to force the obstruction. Each time this occurs, the capillary pressure rises correspondingly. Relief may come to the eye either (1) by the opening up of fresh channels for the escape of the intra-ocular fluid, or (2) by a diminution of the fluid secretion, brought about by the fact that the levels of the vascular pressure and of the intra-ocular pressure have become more nearly equalized. If no such succour is forthcoming, what happens to the eye? Step by step the venous pressure mounts upwards, and with it the capillary pressure, until the whole system of vessels within the eye reaches the blood-pressure level of the arteries at their entrance into the globe; then circulation must cease altogether.

The important lesson to learn is that the great factor which dominates every other, and which ultimately decides the level at which the intra-ocular pressure is to stand, is that of the volumetric contents of the eye. This in its turn is determined by the relative rates of secretion and of excretion of the intra-ocular fluid.

CHAPTER IV

THE PATHOLOGICAL ANATOMY OF GLAUCOMA

In our search for the causes of a disease, we turn naturally to a study of its pathological anatomy. In the case of glaucoma this method is very unsatisfactory, as all our material is obtained from globes far advanced in the morbid condition. In such, the changes which *cause* the rise in tension are obscured and overlaid by those which *result from* the condition. Consequently, we are unable to distinguish between cause and effect, and are therefore thrown back largely on clinical observation. The difficulty is heightened by the fact that a large number of widely different conditions are grouped under the heading of glaucoma, because a rise in the pressure within the eye is the leading feature in all alike.

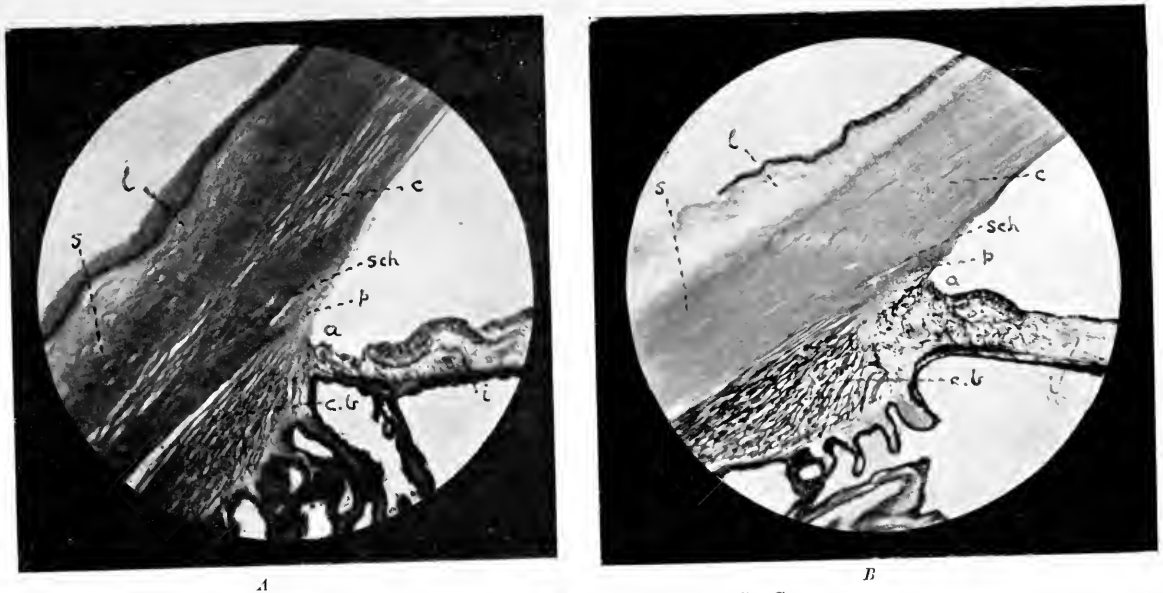
We may dismiss, as unworthy of serious consideration, the view that **changes in the vortex veins** are an important factor in causing the disease. We may also deal summarily with the idea that glaucoma is primarily a **disease of the vessels;** for we now know well that the familiar vascular changes are a result, and not a cause, of the increase in intra-ocular pressure. Again, we can jettison the view that the cupping of the disc is due, not to an increase in the intra-ocular pressure, but to an **active atrophy of the optic nerve itself.** It remains to deal with the suggestion that the cause of the condition is to be sought in a change in the tissues of the eye, brought about by the deposit in them of waste acid

products—with the result that they take up moisture more readily than natural, and swell up in consequence. The evidence available is all against such a view.

Fibrosis of the Pectinate Ligament.—It has been shown that, as life advances, the fibres composing the pectinate ligament become thicker and thicker. This causes them to encroach upon the spaces of the open network of the ligament. It has been suggested (T. Henderson) that this change opposes an obstacle to the free passage of aqueous fluid from the anterior chamber to the canal of Schlemm, since the current finds its way along these open clefts or spaces. Again, the view has been brought forward (A. Thomson) that the changes in the ligament may interfere with the pump action, whereby fluid is supposed to be sucked from the chamber into the canal, since the rebound after each stroke of the muscular (iris and ciliary muscles) action is dependent on the elasticity of the ligament. That both these factors are to be taken into account is accepted, but it must be remembered that the changes in the ligament are said to be *invariably* found as life advances, whilst glaucoma is far from being so widespread as this. They may be taken to be contributory, but no more.

Closure of the filtration angle is caused by the iris base being pressed against the periphery of the cornea. At first the closure is simply mechanical, the two surfaces merely lying in close apposition. Later a plastic exudation is thrown out, which causes the two membranes to become adherent to each other. Later still the iris tissue becomes firmly compressed, and eventually undergoes atrophy. The final stage is a retraction of the ciliary processes which drags the iris back with them, and may even tear through the adhesions between it and the cornea.

During the earlier stages of the process, fluid can still



A B

FIG. 4.—ANGLE OF CHAMBER: *A*, OPEN; *B*, CLOSED.

s, Sclera; *l*, limbus; *c*, cornea; *sch*, canal of Schlemm; *p*, pectinate ligament; *a*, angle of chamber; *c.b*, ciliary body; *i*, iris.

Note that in *A* the angle is widely open, and the pectinate ligament is freely bathed by the fluid of the chamber; whilst in *B* the base of the iris is adherent to the pectinate ligament, thus shutting the canal of Schlemm off from the aqueous fluid.

(*From original photos by E. E.*)

[*To face p.* 14.

find its way along the meshes of the iris, to escape by the drainage channels; but as compression asserts itself such a flow is very severely interfered with. At the same time, the pressure to which the parts are subjected interferes with the circulation of blood, and paves the way for atrophy to follow.

The closure of the filtration angle is brought about by different factors in different cases. In congestive glaucoma, the ciliary processes are enlarged owing to engorgement by blood, and consequently push the iris base forward in front of their expansion. At the same time their forward movement permits the lens to advance, and this, again, in its turn thrusts the iris forward.

In simple glaucoma, the displacement forward of the iris is a result of the enlargement and advance of the lens which take place as life progresses. The action is much slower than that met with in congestive cases, and the tendency to the formation of adhesions is much less. It only remains to add that the changes in the lens just discussed are also operative in congestive cases, but their rôle is then a subordinate one compared with that of ciliary congestion.

The changes we have been describing naturally lead to a **shallowing of the anterior chamber.** The latter is well known to be a leading feature of glaucoma.

The Dimensions of Glaucomatous Eyes.—The eyes of glaucomatous persons are, as a rule, smaller than those of the non-glaucomatous. The measurement of the transverse diameter of the cornea furnishes the best indication of the size of the eye. The factor of real importance is the relationship between the size of the eye and that of its contained lens. The lens grows continuously throughout life, whilst the cornea of the fœtus has nearly attained its full dimensions at birth, and the sclera ceases to grow before the rest of the body does.

Since the lens grows larger and larger as life advances, whilst the globe stands still, it is clear that the older the eye becomes, the more likely is it that the lens will become too large for safety; and, again, the smaller the globe is, the earlier in life will this disproportion occur (Priestley Smith).

CHAPTER V

THE CAUSES OF GLAUCOMA

HAVING reviewed the pathological anatomy of the subject, we now pass on to its ætiology.

1. **Age** takes precedence over all known causes of the disease. In the earlier decades of life glaucoma is rare; its frequency increases slowly in the fourth decade, more rapidly in the fifth and sixth, remains stationary in the seventh, and then diminishes again. **How does the advance of life cause glaucoma?**

(*a*) By bringing about *alterations in the anatomical conditions of the parts.* Amongst such we may class (i.) enlargement of the lens, (ii.) advancement of the lens due to slackening of its zonule, and (iii.) fibrosis of the pectinate ligament.

(*b*) By the influence of *degenerative changes*, which may lead either to an increase in the volume of fluid secreted by the eye, or to a change in the constitution of that fluid making it less suitable for ready filtration.

(*c*) By rendering the eye liable to *auto-intoxication* and all the consequences which flow therefrom.

(*d*) By *interference with* that free and even working of the *vascular system* which is a characteristic of healthy youth.

2. **Sex.**—Congestive glaucoma is nearly twice as commonly met with in the female as in the male. The unstable condition of the female nervous system at the time of the climacteric probably accounts for the disparity.

On the other hand, non-congestive glaucoma is rather more frequent in men than in women.

3. **Heredity.**—The influence of heredity is revealed both in the race and in the family. Jews, Egyptians, and certain races of negroes, are believed to be especially liable to the disease.

4. **Errors in Refraction.**—The idea that the myope is immune from glaucoma, whilst the hyperope is especially liable to it, is a myth. Errors in refraction probably play a very small part in the ætiology of the disease.

5. **Mydriasis.**—Anything that dilates the pupil tends to block up the angle of the chamber, and so, by causing an obstruction to the outflow of intra-ocular fluid, predisposes the eye to glaucoma. The causes of dangerous mydriasis are (i.) the abuse of drugs; (ii.) the exclusion of light from the eye; (iii.) the influence of violent or exhausting emotions; and (iv.) the effects of depressing disease. On this subject one word of warning is demanded. There is *no* drug which produces mydriasis which can be safely instilled into an eye predisposed to glaucoma, unless very careful safeguards are employed.

6. **Nerve Shock and Strain.**—Family bereavement, haunting fear, business anxiety, long fatiguing anxious night-watches, prolonged mental overwork, sleeplessness, and such-like conditions, are often the factors that determine an attack of glaucoma.

7. **Febrile Diseases.**—These may act in one of two ways: (*a*) by exhausting the vital energies of the patient, or (*b*) by a toxic interference with the vascular arrangements of the eye, or with the integrity and efficiency of the secretory and excretory mechanisms of the globe.

8. **Injuries.**—Very slight injuries of the eye or head sometimes determine an attack of glaucoma. *Faute de mieux*, we fall back on the explanation of an interference with the vasomotor mechanism of the eye.

CHAPTER VI

THE DIAGNOSIS OF GLAUCOMA

In the great majority of instances, it is an easy matter to make a correct diagnosis in a case of glaucoma. The danger of a failure to do so does not arise from any inherent difficulty, but rather from the surgeon's forgetfulness of the possible occurrence of the disease in his practice. Difficult cases are undoubtedly met with, and over these even experts may differ; but their very nature is such as to provoke a suspicion of glaucoma in the mind of any practitioner. Under these circumstances, it is not difficult for him to put the responsibility on to other shoulders. The essential point is that he should be forewarned, and therefore forearmed. Every patient he meets with failing vision should make him think of the possibility of simple glaucoma. Every case of severe headache he is called in to treat should arouse in his mind a suspicion of congestive glaucoma, especially if it is accompanied by vomiting.

Early diagnosis, always important, has become much more so since modern methods have made the disease easier and safer to handle. Congestive glaucoma is a surgical emergency, whilst the simple disease is a dangerous pitfall of Medicine.

There is no such thing as prodromal glaucoma. The disease must be divided into three stages: (1) the early, the time of election for treatment; (2) the established, when decisive action is urgently called for; and (3) the late, often, alas ! too late.

Glaucoma is divided into two classes: (*a*) the simple, or non-congestive, which presents no evidence of marked vascular disturbance; and (*b*) the congestive (incorrectly termed "inflammatory"), in which an obstruction to the efferent veins at their points of exit from the eye causes a damming up of blood within the organ. A case may be simple throughout, or congestive throughout, or the one form may pass into the other and back again into that from which it first sprang. We may even meet with simple glaucoma in one eye, and with the congestive condition in its fellow. The essential feature, however, in all cases alike is an obstruction to the outflow of fluid from the interior of the eye into its excretory channels. Whether a congestive element enters into the case is largely a matter of chance, in which the anatomical configuration of the organ, defective development of the excretory channels, and the suddenness of the onset of the changes which obstruct excretion, all play a part.

The Clinical Course of an Attack of Simple Primary Glaucoma.—The onset is gradual, the signs of congestion are absent, there is no pain and little subjective evidence of any kind; but the visual field shrinks, so that the patient complains that he feels as if he were looking down a tube (tube-vision), and therefore is unable to avoid objects with which he meets. Visual acuity often falls, and cupping of the disc is soon well marked. We may speak of these as the essential triad of glaucoma signs, and put them shortly as (1) contraction of the field, (2) cupping of the disc, and (3) failure of visual acuity. So gradually may the symptoms come on that the patient may only discover their existence when the first eye is lost entirely, and the second has begun to fail. In the great majority of cases the pupil is dilated and sluggish. Later still it is immobile, and surrounded

by only a narrow margin of iris; rarely it may be of normal size until quite late in the disease. Sometimes moderate or even good central vision is retained for a long time. An increase in the tension of the globe is undoubtedly present from the first dawn of the disease, but it is not always recognized, and this for two reasons—viz., (1) that an increase in the intra-ocular pressure may be intermittent, and therefore not always in evidence; and (2) that, as compared with congestive glaucoma, the rise in tension is quite moderate in early cases, and may escape detection by digital examination, though it will be revealed by the use of the tonometer.

It cannot be too strongly impressed on the practitioner's mind that a diagnosis of glaucoma should not be made on any one sign alone. Every possible scrap of evidence, historic or clinical, should first be gathered; a broad view should be taken, and judgment should then be formed. In the early stage a decision may be impossible; careful watching of the patient is then indicated. Later, when glaucoma is established, the most ordinary care suffices, whilst later still no medical man could mistake the condition for anything else.

The Clinical Course of an Attack of Primary Congestive Glaucoma.—A pronounced attack of congestive glaucoma may begin without warning of any kind. More often it is heralded by the appearance of the so-called "prodromata," or is engrafted upon and complicates an attack of apparently simple glaucoma. Such variations, of course, are very numerous.

In most cases, the patient has observed that from time to time his sight has become misty for short periods, generally for a few hours at a time. With this has been associated some degree of pain in the eye, and headache, together with the appearance of rainbow rings around bright lights viewed in the dark. Flashes of light are

frequently seen before the eyes, and are variously described as resembling summer lightning, rolling balls of light, sudden flashing luminous points, fireflies, etc. During the attacks the pupil is dilated, the eye congested, the anterior chamber shallow, and the cornea steamy. Exhaustion, sleeplessness, fatigue, or worry, bring on the crises, whilst rest, food, warmth, and sleep, cause them to pass away. They are frequently noticed either in the early morning, or in the late afternoon or evening. Sooner or later the early condition passes into the phase of established glaucoma. The transition from one to the other may be gradual and imperceptible, or, on the other hand, the new phase may be ushered in by a crisis, whose severity is such that the patient, quite erroneously, dates the onset of his disease from it. The leading symptoms are—(1) severe trigeminal neuralgia, usually referred to the eye and forehead, but sometimes to other parts as well; (2) a rapid fall in vision; and (3) a marked contraction of the visual field. The eye is very congested, as also are the lids. Chemosis may be present. The cornea is steamy and insensitive; the pupil is widely dilated and often oval; the anterior chamber is shallow; the iris appears discoloured; and the lens has a peculiar green look (γλαυκός = sea-green). The eye is very tender and painful, and is felt to be extremely hard. After a varying period the attack passes off, leaving the eye permanently damaged and in a condition of chronic glaucoma, usually of a congestive type, though almost all the signs of interference with the blood-stream may pass away. The downward path of the eye is punctuated by the more or less frequent occurrence of congestive exacerbations of the disease. It will thus be seen that the early attacks may pass gradually into the more severe ones, or the latter may be the first to make their appearance. The eye is gradually drifting towards

an established condition of chronic congestive glaucoma. The visual acuity is impaired; the field of vision is contracted; the tension is above normal; circumcorneal congestion, with distension of the perforating vessels, is present; the cornea readily clouds over, and is losing its sensitiveness; the anterior chamber is very shallow; the pupil is wide and oval, and acts sluggishly; the iris is changing in colour, and atrophic patches are appearing. If the media are clear, we can see in the fundus (*a*) the cupped disc surrounded by a ring of atrophy, (*b*) the constricted arteries, (*c*) the congested veins, and (*d*) the vascular pulsation, all of which are characteristic of glaucoma.

Finally we come to the "late stage of glaucoma," often spoken of as that of "absolute glaucoma." All sight is lost, though the patient may protest that at times he sees as well as ever, a delusion to which the name "memory sight" may be applied. The green reflex, the stone-like hardness of the eye, the degenerative changes which mar the cornea, and the porcelain-like colour of the sclera, with the dilated vessel standing out on its surface, complete the picture already drawn of the earlier stage. Even now there are possibilities of harm to be added, since the patient may suffer from "blind painful eyeball," from perforation of the globe, or even from panophthalmitis.

Let us pause for a moment to lay emphasis on certain points in connection with this disease, be the type what it may.—

1. It is bilateral, and therefore complete blindness is threatened.
2. In the vast majority of cases, it is relentless and progressive.
3. Its keystone is a rise of pressure within the eye.
4. Its fundamental causes are protean.

5. One and all of them act by upsetting the balance between the secretion and excretion of the intra-ocular fluid.

6. The entry of the congestive factor into the drama is an accident, though one of the very gravest proportions.

CHAPTER VII

THE SIGNS AND SYMPTOMS OF GLAUCOMA

We shall take the structures of the eye *seriatim*, and consider the evidence of glaucoma afforded by each.

The Conjunctiva and Sclera.—The only change seen in simple glaucoma is a slight enlargement of the anterior ciliary vessels, especially where they perforate the sclera.

In congestive attacks, the condition present may vary from slight circumcorneal congestion to intense reddening, and even to chemosis. In the intervals between attacks, a permanent enlargement of the anterior veins is always left.

In late glaucoma, the sclera has a bluish-white, porcelain-like appearance, against which the distended anterior ciliary veins stand out in marked contrast.

The Cornea.—There are two symptoms occurring from the earliest stage of congestive attacks—viz., (1) *mistiness of vision*, and (2) *haloes round lights*. These are due to two causes: (*a*) an overstretching of the cornea (the result of an increased intra-ocular pressure), which interferes with the refraction of the membrane; and (2) an actual œdema of the corneal layers, the result of an obstructed circulation.

The mistiness is often seen in the mornings, and gives the erroneous impression of a fog or of smoke in the atmosphere. The haloes round lights are best seen at night, and are clearest around yellow flames. At least two colours are seen, an inner blue and an outer red.

Anæsthesia of the cornea becomes increasingly marked as glaucoma progresses. A *corneal haze* makes its appearance in some cases, and in late stages permanent opacities of the cornea are found.

The Anterior Chamber is always *shallow* during congestive attacks; later it becomes permanently so. The depth of the chamber can best be estimated by looking at it obliquely from the opposite side, under good illumination, with a loupe or an ophthalmoscope. The contents of the chamber are often turbid in congestive cases.

The Iris.—*Dilatation of the pupil* is found in most cases of glaucoma. Exceptions are rare. It is usually well marked. The pupil is often *oval* in form, with the *long axis vertical.* In congestive cases the iris may be discoloured. At a late stage atrophic patches appear in it, through which the red reflex can be seen on ophthalmoscopic examination. In the very late stages, ectropion of the uveal pigment appears, giving the edge of the pupil a velvety black line.

The Ciliary Body.—Weakness in accommodative power, shown by an abnormal *increase in presbyopia,* is the earliest evidence of involvement of the ciliary body. It is an important point, since the need to change his glasses soon may be the first thing to drive a glaucoma patient to seek medical advice. Later a *congestion of the circumcorneal zone* shows an engorgement of the ciliary body with blood.

The Lens.—Owing to the advance of the lens system, as the anterior chamber shallows, a tendency to *myopia,* or to lessening of existing hyperopia, is in evidence. The weakening of the ciliary muscle, at the same time, tends to bring about presbyopia, as above indicated. These factors act in opposition, but the latter is the stronger and therefore the prevalent one.

A form of *cataract* not infrequently complicates glau-

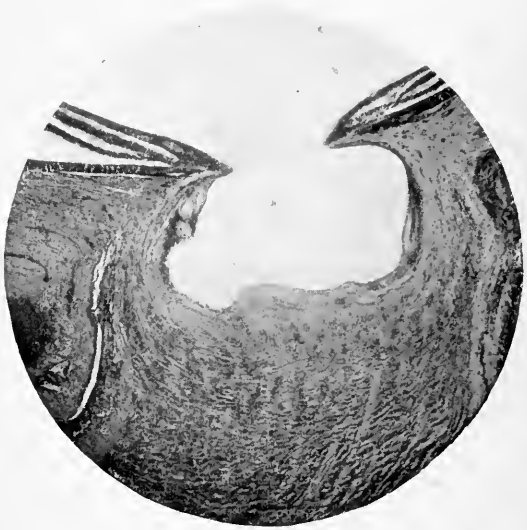


FIG. 5.—CUPPING OF THE OPTIC DISC (GLAUCOMATOUS).

Note (1) the sharp scleral spur on the right side; (2) the overhanging edges; and (3) the retinal vessels hiding under the latter. ×20. (From micro-photograph kindly supplied by Mr. Chesterman.)

[*To face p.* 27.

coma. It has to be distinguished from cataract occurring as an accidental complication of the condition. In some cases cataract is the first morbid condition present, and the glaucoma is secondary to it. The history and appearances of the eye will decide which condition is present.

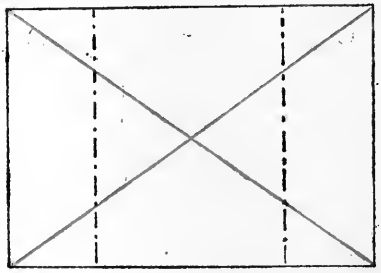
The green reflex in the pupil, which has given glaucoma its name (from γλαυκός = sea-green), is produced by the dilated pupil, combined with some want of transparency in the lens, in the aqueous humour, and in the cornea.

The Optic Nerve and Retina.—The changes found are—(1) *pallor of the disc*—this is partly due to a constriction of the blood-supply, and partly to advancing *atrophy;* (2) a depression of the floor of the disc, the so-called *cupping of the disc:* (3) an *alteration in the apparent direction of the vessels* as they pass from the disc on to the surrounding fundus; (4) the presence of *pulsation* in the retinal vessels; (5) a *change in the size* of the retinal arteries and veins.

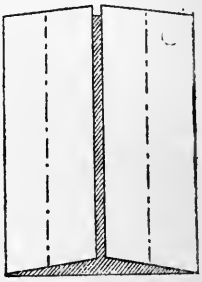
The *cupping of the disc* is the best-known and most easily detected sign of the disease. It is due to the nerve entrance being the weakest spot in the globe, and therefore yielding first under the increased pressure within the organ. The important points to grasp are, (*a*) that the whole of the disc is depressed in the great majority of cases; (*b*) that, as each vessel passes from the disc on to the retina, it shows a kink as it bends over the lip of the cup; and (*c*) that some of the vessels when traced from the floor of the disc on to the retina seem to change their direction somewhat, and so to emerge parallel with what might have been expected to be their course. The last-named phenomenon is frequently ill understood by students, but is very easily made clear by the aid of the following simple device.—

Take a half-sheet of note-paper, 7 inches long by 4½

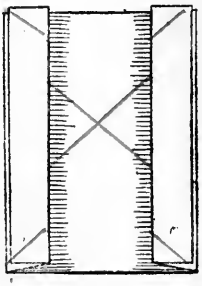
wide (the exact dimensions are unimportant), and join the corners by drawing two diagonal red lines (Fig. 6, A), thus making a multiplication sign, whose four arms are



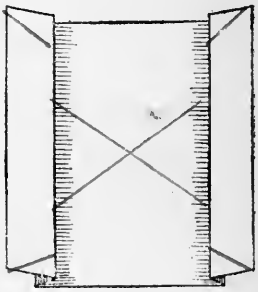
A.—The red lines are to represent blood-vessels. The dotted lines indicate where the paper is to be folded the first time.



B.—The first folding of the paper shown complete; the dotted lines show position of the second fold.



C—The second folding of the paper shown completed.



D.—The double flaps raised in concertina fashion, to show the overhanging edge of the disc and the apparent alteration in the direction of the vessels, as they emerge, on the plane of the retina.

FIG. 6.

designed to represent four emerging vessels. Fold each edge of the paper forward towards the centre, so that the two edges meet each other over the centre of the

multiplication sign (Fig. 6, B). Take each edge in turn and fold it backward to meet the first folds (Fig. 6, C). Then raise the two double flaps so made, forward in a concertina-like fashion, and look down on the device from above (Fig. 6, D). The unfolded part of the paper, lying flat on the table, diagrammatically represents the floor of the disc; from the first fold to the second on each

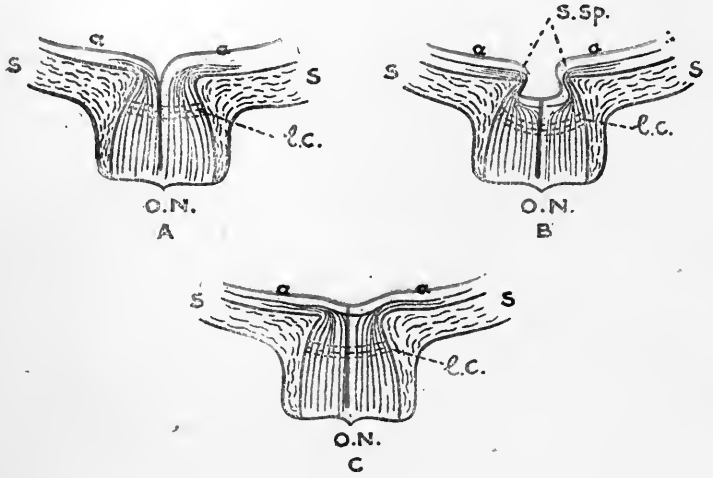


FIG. 7.

A, physiological cup; *B*, glaucomatous cup; and *C*, cup of optic atrophy (modified from Fuchs). *O.N.*, Optic nerve; *l.c.*, lamina cribroso; *S.*, sclera; *S.Sp.*, scleral spur, which is supposed to damage the retinal fibres, as they bend over it; *a*, *a*, retinal artery.

side represents the shelving wall of the disc; from the second fold to the free edge represents the fundus level. The apparent break in the continuity of the straight lines originally made is very striking, and aptly illustrates our point.

In late cases, the retinal vessels are sometimes seen to be drawn over to the nasal side, in what looks like a leash.

Pulsation of the Retinal Vessels.—If the veins alone pulsate, little stress need be laid on the phenomenon; if, however, this pulsation is strong, and if a light pressure on the globe with a forefinger causes the arteries to pulsate as well, or if the arteries pulsate even when the globe is untouched, there is a strong suspicion of a rise in intra-ocular pressure.

A Change in the Size of the Vessels.—The arteries tend to become more constricted, and the veins larger and fuller than natural.

Caution.—A glaucomatous cup must be distinguished from a physiological cup, from an atrophic cup, and from a coloboma of the optic nerve (Fig. 7).

The physiological cup is rarely deep; it only involves a part of the disc, usually the outer quadrant; some at least of the vessels pass out without a kink; the main part of the papilla is of normal colour.

The atrophic cup is very shallow, the disc is grey, and the vessels are not kinked.

In coloboma the apparent disc is much enlarged and the cup is of great depth. The history of the case may be of value.

The Retina is starved of arterial blood, and the outflow of venous blood is obstructed; it is also subject to direct pressure; the circulation through the choroid too is impeded, and consequently the outer layer of the retina has its nutrition interfered with. The result is a limitation of the visual field, and the production of subjective sensations of light.

The Size of the Eye should always be noticed in glaucomatous cases. It can best be judged of by measuring the diameter of the cornea, which will often be found to be below normal. The average horizontal diameter of the cornea in healthy subjects is 11·6 mm. (Priestley Smith).

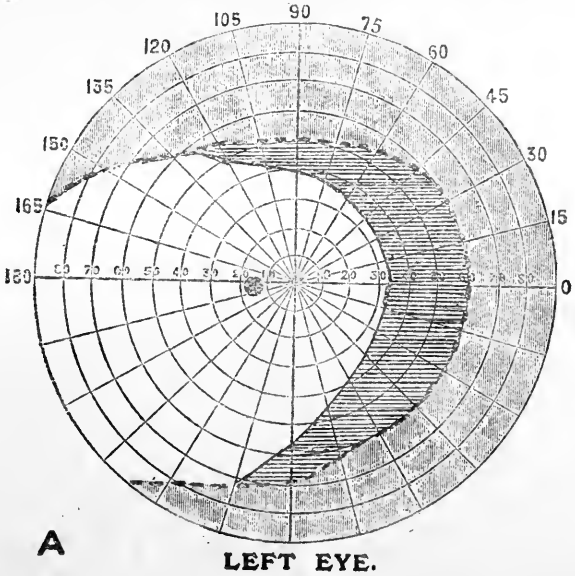
Pain varies enormously in different cases, and is usually a measure of the congestion present. A simple glaucoma may be painless throughout, whilst an acute congestive attack is marked by terrible suffering. There is severe neuralgia not only in the eyes, but also in the head, teeth, ears, and neck. The agony may be so great as to lead to vomiting and pyrexia. In less acute cases, the pain and headache are intermittent, or may be more or less constant. Sleep, food, or rest, often relieve the pain.

Photopsiæ, or subjective sensations of light, are due to interference with the retina. They may occur in other affections of the retina and choroid, in some cases of cerebral disease, in megrim and other nervous affections, in neurasthenic patients, under conditions of cardiac syncope, and in errors of refraction. In glaucoma they are worse when the patient first lies down, and are relieved after rest as the night advances, whilst in cerebral cases they are often most troublesome later in the night.

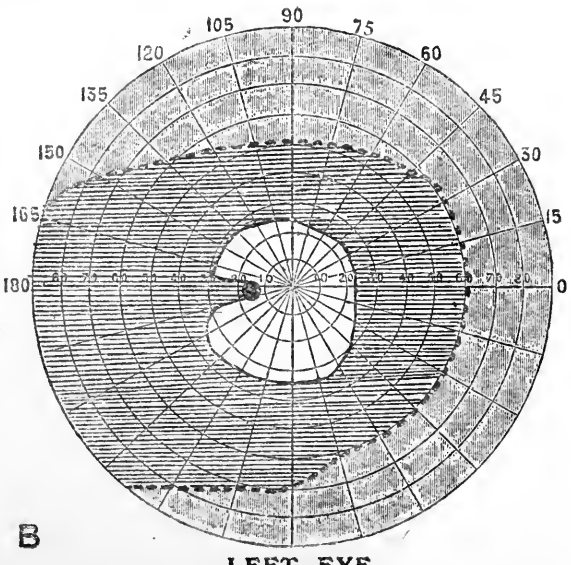
Rainbows round lights have been already spoken of. It remains to add that similar phenomena may be observed in conjunctivitis owing to flakes of mucus on the cornea; in some cases of early cataract; in eyes undergoing various forms of drug treatment (*e.g.*, painting with solution of silver nitrate); and habitually in the eyes of certain apparently normal people.

A Diminution of Visual Acuity may be due to lesions of the cornea, to opacities in the aqueous fluid, in the lens, and in the vitreous, and to atrophy of the optic nerve and retina.

The **Visual Field** is early affected, and affords one of the most delicate tests of the presence and advance of glaucoma. It undergoes a characteristic contraction, the nasal portion usually suffering first (Fig. 8A). The colour fields are but little affected; central vision is usually retained till quite late. The most delicate test of



A, The Visual Field in an early case of glaucoma taken with a large (5 mm.) white object at a distance of 33 cm. This is the ordinary method of perimetry.
Notice the loss of the nasal side of the field.



B, The Visual Field in glaucoma mapped out by means of a small (1 mm.) white object at a distance of 1 m. This is Bjerrum's method. Notice that the blind spot is continuous with the area of lost field on the one hand, and with that of still preserved vision on the other. (Bjerrum's sign.)

FIG. 8.

all is that devised by Bjerrum. It consists of examining the field with a very small object (usually 1 mm. in diameter) at the relatively great distance of 1 metre or even 2 metres. When so tested, it is found that the visual field is much smaller than the normal, and that the scotoma due to the blind spot is much enlarged, and is continuous in one direction with the loss of the peripheral field (Fig. 8B). Paracentral scotomata are also observed, and are considered to be of considerable importance in diagnosis.

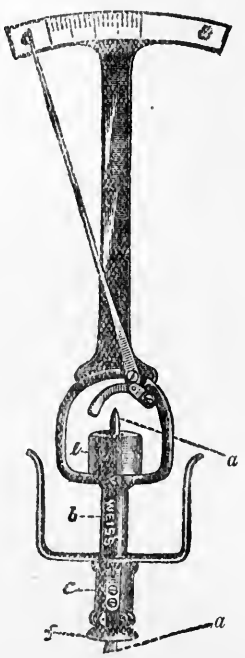


FIG. 9.—THE SCHIÖTZ TONOMETER.

a a, sliding rod on which *l*, the weight, is fixed; *b*, a hollow cylinder in which *a* slides; *c*, the collar on which *a* moves by wheel bearings; *f*, foot-plate which rests on the eye. The rod *a* actuates the lever above it, and so records on the scale on the arc.

Tonometry.—The old method of finger tonometry will always retain its place in rough-and-ready work, but is being steadily and rapidly replaced amongst ophthalmologists by the use of the Schiötz tonometer, or of some modification of that instrument. Mechanical tonometry, when carefully carried out, is of the greatest value. It is, however, a method for the specialist rather than for the general practitioner.

DIFFERENTIAL DIAGNOSIS

A case of *simple or non-congestive glaucoma* may be mistaken for cataract, for an error in refraction, or for an affection of the retina and choroid or of the optic nerve. A careful examination of the case will usually clear the matter up at once; if not, it must be

kept under observation for a while, when the evolution of the cupping of the disc and of the changes in the field will settle the question. The point to insist strongly upon is that in all such cases a routine examination of the media, fundus, fields, and tension, is urgently demanded.

Congestive glaucoma, on the other hand, is often mistaken for conjunctivitis, for iritis, or for "bilious" or other headache.

A case of conjunctivitis should easily be distinguished from one of congestive glaucoma, since in the latter we find—(1) marked circumcorneal congestion; (2) steaminess of the cornea; (3) dilatation of the pupil; (4) a considerable rise in tension; (5) severe headache and neuralgia. None of these signs are usually present in eyes suffering from conjunctivitis.

In iritis the pupil is contracted, and the loss of pattern and the change in colour of the iris are prominent features, whilst the tension is not as a rule raised.

The diagnosis from bilious headache, gastric catarrh, and other conditions associated with headache and vomiting, can always be easily made by any surgeon who will trouble to examine the eyes. So long as it is borne in mind that these cases may possibly be glaucomatous, there is not the least danger of a mistake in diagnosis; the latter can only arise when the surgeon is taken off his guard.

CHAPTER VIII

THE TREATMENT OF GLAUCOMA

The treatment of glaucoma falls naturally under two headings: (1) the general and meiotic, and (2) the operative. These will be dealt with in turn.

The General and Medicinal Treatment of Glaucoma.—The patient, who is known to be suffering from glaucoma, should be carefully warned to regulate his life in such a way as to avoid, as far as possible, anything which may provoke a vascular storm in the eye. A list should be given him of the following sources of danger: constipation, overfatigue either mental or bodily, anxiety and business worry, sleeplessness, exposure to chill, and any form of violent mental excitement. The use of any form of mydriatic drug is strongly contra-indicated, unless subsequent meiosis can be assured. Even the instillation of cocaine may be dangerous. If it is deemed necessary to use one of the milder mydriatics for any purpose, the patient must not be allowed to pass away from supervision until the pupil has contracted again under the influence of a meiotic. It is a wise precaution to provide the patient with a further supply of weak eserine or pilocarpine drops, to be used should the pupil tend to dilate again in the course of the day.

It is not enough to give general warnings. We must never forget that the glaucomatous eye is a "sick eye in a sick body." Every system of the body should be overhauled with the thoroughness of an examination for life insurance, special attention being paid to the vascular

and urinary organs. Any source of auto-genetic poisoning should be eliminated, and the diet and habits should be carefully regulated. Alcohol should be forbidden, or at least strictly limited. It is to be remembered that infectious diseases, and amongst these we may especially mention influenza, carry additional dangers to the victims of glaucoma.

Meiotics.—There are two entirely distinct conditions under which meiotic drugs are used; these are—(1) in the congestive phases of glaucoma, to restore the eye to its normal vascular condition, or at least to as near to that as may be possible; and (2) in the absence of congestion, as a substitute for operative treatment. We will take these points in order.

In congestive attacks, the action of 1 per cent. solutions of eserine or of pilocarpine should be boldly pushed, the drops being instilled every few hours until a result is obtained. With this should be combined free leeching, active purgation, rest in bed, milk diet, and the administration of morphia (unless the last-named is otherwise contra-indicated).

If it is decided to employ meiotics over a long period in chronic congestive cases, pilocarpine is preferable to eserine, and the weakest solution that will keep the pupil contracted should be used. As time goes by, the strength of the drops may require to be augmented. To begin with, half a grain to the ounce will probably suffice. Care must be taken to counteract the tendency of these drugs to produce chronic catarrh of the eye, and the patient must be warned never to let his stock get stale or run out. In this connection there are certain important warnings to be given.—

1. The treatment is merely palliative, not curative.

2. Meiotic treatment is useless in simple glaucoma. Its only rôle is in cases of the congestive type.

3. The glaucoma patient should be kept under constant observation, careful periodic examinations being made of the field of vision, of the tension of the eye, of the cupping of the optic disc, and of the visual acuity. The above order is deliberate, and signifies the relative importance to be attached to the four points. Should the condition of the eye be deteriorating, operative treatment should be substituted without delay for the medicinal regimen. The latter has sometimes been spoken of as "expectant treatment." If the patient is losing ground, it can be expectant only of disaster.

4. Every authority on glaucoma from von Graefe onwards has laid stress on the fact that, if an operation is to be performed for the relief of glaucoma, the earlier it is done, the better is the chance of success. Hesitation is dangerous. Once we see that other measures are failing, we must recommend and carry out an operation with the least possible delay.

5. The exhibition of meiotics can only have a rational basis so long as the contractility of the iris is unimpaired. Therefore in late cases it is useless.

Massage.—The great value of massage in glaucoma does not appear to be sufficiently appreciated. The tension of an eye can be quickly and sensibly reduced by digital manipulation, and the patient can be taught to perform the simple movements for himself. These are two in number. The first consists in placing the tips of the two index fingers on the upper lid over the globe (the surgeon standing behind the patient), and pressing with each finger in turn in the direction of the centre of the eye. The movements should be slow at first, and made more rapidly later, as the operator acquires skill and the patient toleration; the subject must look downward, and gently close the eyes. The period of massage should be short at first (about half a minute), and should

be extended until it lasts three to five minutes, thrice daily or more often. The second movement is a gentle circular, smoothing action performed with three fingers of one hand, through the gently closed lids. It serves the same purpose as the wide smoothing movements used after massage of other parts, and only needs to be done for about fifteen or twenty seconds.

Massage of the eyes should be just as much a routine measure in the treatment of glaucoma as the use of meiotics or the regulation of the patient's habits. Not only so, but it is also a measure of very great value in augmenting the effect obtained by operative measures, when that proves to be on the short side.

The Operative Treatment of Glaucoma.—We begin with a short description of the various operative procedures before the profession, leaving the reader to refer for details to more lengthened treatises on the subject.

Iridectomy.—This operation, introduced by von Graefe, held sway for nearly fifty years, until its supremacy was challenged by the procedures which aimed deliberately at the formation of a filtering scar—*i.e.*, a scar which allows the aqueous fluid to escape indefinitely from the interior of the eye to the potential subconjunctival space on its surface.

The removal of a portion of iris may relieve a glaucomatous patient in several ways.—

1. If performed quite early, before any adhesions have formed between the iris base and the cornea, it establishes a limited space over which the normal filtration can continue unimpeded either by dilatation of the iris or by the formation of adhesions over the area of the coloboma.

2. The pressure within the eye is temporarily relieved. The ocular circulation is permitted to resume its normal character, and the patient, warned by the severe lesson he has received, may so alter his life habits as to protect

himself from another attack for a long time to come. The predisposing factors of glaucoma are ever with him, and are steadily, if slowly, increasing in their power for harm, but, thanks to his care, the exciting causes are as far as possible eliminated, and so an attack is avoided.

3. A filtering cicatrix may be accidentally established as a result of the operation, though this is not what was deliberately aimed at.

Certain plain lessons are here to be learnt.—

(*a*) If iridectomy is to be useful, it must be done at the earliest possible stage of the disease.

(*b*) The incision must be peripheral, so as to admit of the iris being removed as far back as possible.

(*c*) The incision must be large, so that the portion of iris removed shall be as wide as possible; this insures a wide area of the angle for free drainage.

(*d*) If we desire to produce a filtering scar, we should employ an operation deliberately designed for the purpose, and not one which causes it only by accident.

We shall now pass on to consider the various procedures which aim deliberately at the production of a filtering scar, but before doing so we must make it quite clear what we mean by this term. A **filtering scar** is one which allows the intra-ocular fluid to pass through the tunic of the eye into the subconjunctival tissue, and there to be taken up by the lymph and blood vessels. The ocular evidence of such a scar is a boggy or œdematous condition of the overlying conjunctiva. When a probe is pressed over this area, marked pitting takes place. It has been pointed out that a similar pitting may be demonstrated in some normal, and in not a few inflamed, eyes. It is necessary, therefore, to emphasize the point that the pitting over a true filtering scar is more marked, and usually much more fluid, than that

in the conditions above mentioned. The diagnosis rests on the degree and fluidity of the œdematous condition.

An effort has been made to distinguish between a "filtering scar" and a "fistulous scar." There is, however, no anatomical basis for the view that it is possible to establish a spongy filtering condition of such a cicatrix, and it is safe to assume that all cicatrices supposed to be of a spongy nature are in reality permeated by tiny fistulæ.

Lagrange's Operation.—Lagrange was the first to remove a portion of the ocular tunic (sclerectomy) in order to make sure of establishing a filtering scar. The following are the steps of the operation.—

1. A small shelving corneo-scleral flap is cut with a Graefe knife, and a wide conjunctiva apron is added thereto (Fig. 10A).

2. The conjunctival flap is turned down, and the corneal lip of the wound is removed with scissors (Fig. 10B).

3. Iridectomy is performed, if required (Fig. 10C).

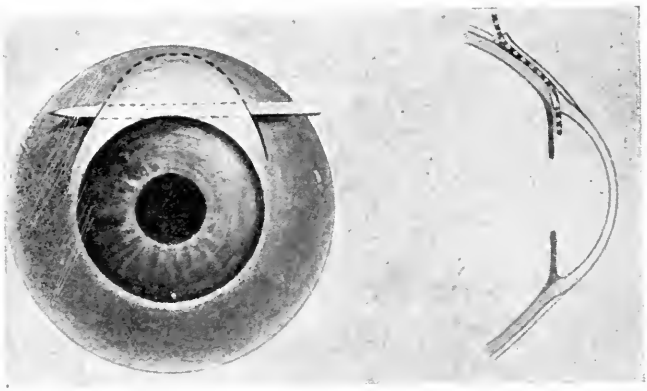
4. The flap is stroked into position and the wound closed.

The result of the operation may be seen in Fig. 10D.

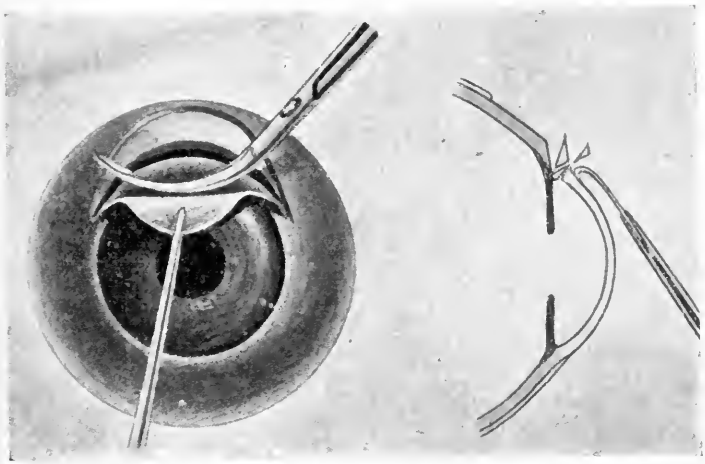
Herbert's Operations must be spoken of in the plural, as the exact procedure has varied from time to time. The underlying idea of all of them has been to obtain a filtering scar by a modification of the operation of sclerotomy. We shall take the procedures *seriatim*.

1. His first attempt (1906) was to make the lips of a small sclero-corneal incision as jagged as possible, in order to prevent primary union, and so to obtain a permanently weak scar.

2. In 1907 he introduced the wedge isolation operation, the object of which was to isolate a wedge of sclera close to the limbus, and to leave it *in situ*, in the hope that, being cut off from its blood-supply, it would shrink, and



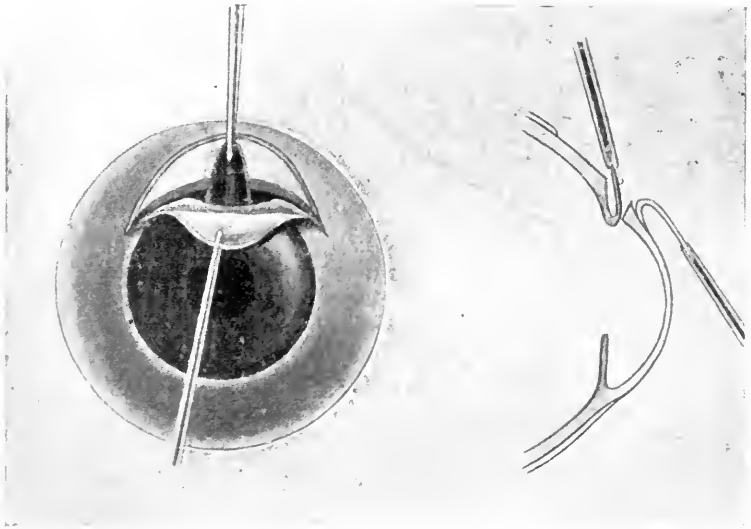
A.—Section of the Sclera and Conjunctiva.



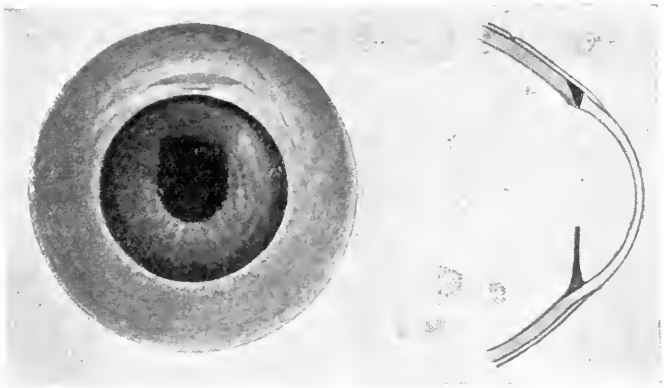
B.—Resection of the Sclerotic.

FIG. 10.—ILLUSTRATING LAGRANGE'S OPERATION (*A*, *B*).

[*To face p.* 40.



C.—The Making of the Iridectomy.



D.—The Result of the Operation.

FIG. 10.—ILLUSTRATING LAGRANGE'S OPERATION (*C*, *D*).

[*To face p.* 40.

so allow of filtration from the interior of the eye into the subconjunctival space around its margins.

3. Later he introduced his small flap operation, which consisted in the making of a small keratome incision from behind the limbus into the chamber, and then carrying two or four cuts forward at right angles to this, in order to form a trap-door flap of sclera. The cuts were made with special scissors.

4. Latterly he appears to have gone back to a procedure that he favoured in his earliest efforts—namely, the attempt to establish a fistula by tucking conjunctival tissue into a scleral wound close behind the limbus. The tuck is kept in place by a metal plug, sutured in position and kept there for some days following the operation.

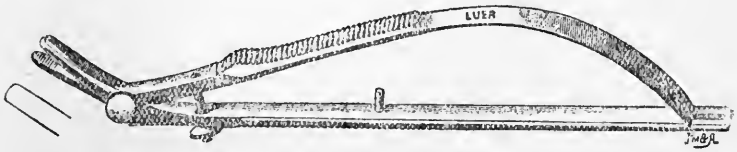


FIG. 11.—HOLTH'S PUNCH FORCEPS.

The drawback to all these procedures is that, like their prototype sclerotomy, they are apt to fail owing to healing up of the wounds made. On the other hand, it is only fair to add that Herbert's contention is that the modern operations, which remove a definite piece of sclera, are apt to do too much.

Holth's Operation.—A sclero-corneal flap is cut with a keratome, a conjunctival apron being fashioned by entering the knife into the conjunctiva some distance above the point of scleral puncture. Iridectomy follows, and then the anterior lip of the wound has a tongue cut out of it by means of a specially designed punch. The flap is replaced in position.

Fergus's Operation.—A conjunctival flap is dissected

up to the cornea, but not into it; a disc of sclera is cut out with a 3 mm. trephine, close up to the limbus; in a certain number of cases a cyclo-dialysis is added; the ciliary body is laid bare in the wound. Some stress is laid on the opening up of a communication between the anterior chamber and the suprachoroidal space.

Sclero-Corneal Trephining (the author's operation) has sometimes been confused with the last-named operation, with which it has little in common, beyond the fact that

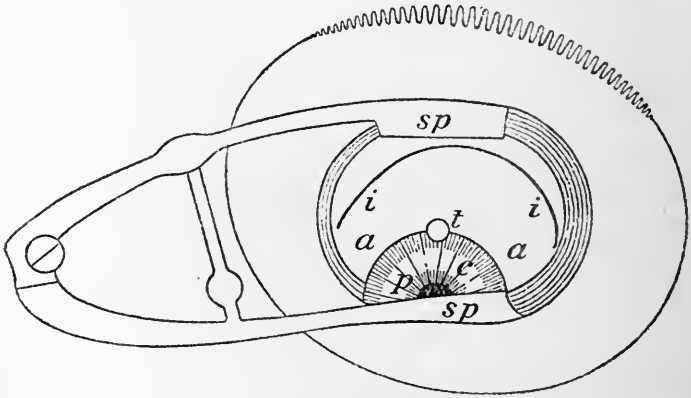


FIG. 12A.—SCLERO-CORNEAL TREPHINING.

sp, speculum; *ii*, incision in conjunctiva; *c*, cornea; *t*, trephine-hole; *p*, pupil; *aa*, uncut conjunctiva, which serves to allow filtering fluid from the neighbourhood of the trephine-hole to pass into the rest of the subconjunctival space.

a trephine is used in both of them. The object of the procedure is to tap the anterior chamber, to drain it permanently into the subconjunctival space, and, in doing so, to avoid if possible any interference with, or impaction of, the uveal tract. An incision concentric with the cornea maps out a large conjunctival flap, which is dissected down to the limbus, the whole thickness of the tissue being taken up. The dissection is carried on into the substance of the cornea for another millimetre,

the cornea being actually "split" over this area. This flap being held down out of the way, a 2 mm. trephine blade is next applied to the corneo-scleral margin, and a disc is cut out in such a manner that it remains hinged on the scleral side. At the same time the iris protrudes

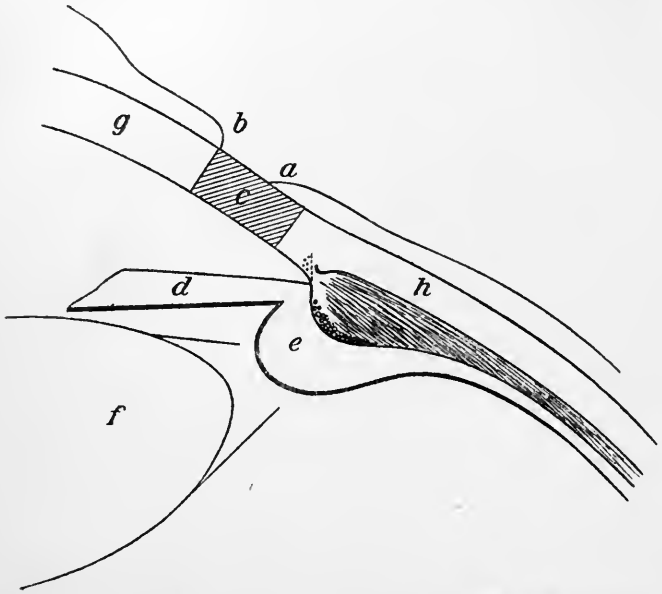


FIG. 12B.—DIAGRAM TO SHOW IN SECTION THE RELATION OF THE PARTS IN A TREPHINED EYE.

a, normal position of conjunctiva; *b*, conjunctiva reflected on to the cornea, after "splitting" of that membrane; *ab*, area of the crescent formed by "splitting" the cornea; *c*, the piece of sclero-cornea removed by the trephine (it is shown shaded); *d*, iris; *e*, ciliary body; *f*, lens; *g*, cornea; *h*, sclera.

through the opening. The disc and iris are seized in one grip of a pair of iris forceps, and cut off with a single snip of the scissors. The iris is replaced, and the conjunctiva is smoothed into position and sutured there. The advantages claimed for this procedure are as follows.—Its technique is not difficult; the sclerectomy on which

drainage depends is the smallest possible, and is made on an intact globe; consequently the dangers of vitreous prolapse and of intra-ocular hæmorrhage are reduced to a minimum; finally, the operation can be repeated, if need be. The accompanying illustrations (Figs. 12A, 12B) serve to make the steps of the operation plain. Full details of the method are to be found in the author's book on the subject.*

Thread Drainage.—A number of operations have been put forward in which a thread is introduced into the anterior chamber, and its ends are buried in the sub-conjunctival tissue. It has been supposed that they would favour the spread of drainage under the flap. If any of the modern operations are carefully performed, there will be little difficulty in the diffusion of the drainage fluid. On the other hand, the introduction of a piece of thread, in free communication with the interior of the eye, appears to be a needlessly dangerous procedure. Recognizing this risk, Prince has substituted a gold drain for the thread. If a drain is to be used at all, this is certainly a preferable expedient.

Cyclo-dialysis, or the separation of the ciliary body from its attachment to the sclera, was designed to open up a communication between the anterior chamber and the suprachoroidal space. It is now very seldom resorted to.

Two subjects remain to be discussed:—(1) the operation to be selected in any individual case; and (2) the arguments that can be used against the undertaking of any operative procedure.

1. **The Selection of an Operation.**—There is at the present time the greatest diversity of opinion amongst

* "Sclero-Corneal Trephining in the Operative Treatment of Glaucoma" (second edition, 1914), by Lieut.-Colonel R. H. Elliot. London: George Pulman and Sons, Ltd.

experts on this subject. There are those who would perform iridectomy in all cases; there are others who would always resort to one or other of the forms of filtering operations; there are still others who would select the operation according to the case. Much depends upon the idiosyncrasy of the surgeon and on his past experience. There is no rule to be laid down. The practitioner will be well advised to leave such technical matters to the judgment of the surgeon he calls in. At the same time it should be said that there are a very large number of surgeons who hold to the broad general rule to perform iridectomy in acute and subacute cases, and a filtration operation in chronic ones. The author's personal view is that sclero-corneal trephining is the safest, the easiest, and the simplest operation in any and every case of glaucoma, with two exceptions. These are (1) glaucoma secondary to the swelling of a maturing cataract, and (2) that which follows any condition which places the aqueous and vitreous chambers in free continuity with each other. He can claim that this view is founded on a large practical experience, and that it is supported by that of a number of very able surgeons; at the same time, he would freely admit that many dissent from it.

2. **The Arguments against Operation.**—These are four in number:

(i.) There are a certain number of cases of glaucoma on record in which the *status quo* has been maintained for years by medicinal treatment alone. These rare instances have encouraged many surgeons to delay operation, and have caused a loss of sight out of all proportion to the meagre gains which can be laid to the credit of the "expectant treatment."

(ii.) Any operation for glaucoma, at a late or comparatively late stage of the disease, may be followed by

a considerable deterioration in visual acuity, associated with a marked loss of field. The author believes that such cases never occur if operation is undertaken early, and that even in the late stage they are rare in modern filtration operations, as compared with iridectomy. Against this must be set the hopeless prognosis of the very great majority of cases of glaucoma when left to themselves.

(iii.) Much stress has been laid on the bad late results that may follow an operation for glaucoma, and especially on the danger of "late infection." By that term is understood an infection of the eye through the operation wound, long after the latter has healed, and quite independent of the introduction of sepsis at the time of operation. This accident has followed all forms of operative procedure, including iridectomy and sclerotomy, but it is probably more frequent in those cases in which a filtering scar has been deliberately produced. Much may be done to lessen this danger by the use of a proper technique at the time of operation. At the same time it would be idle to belittle the danger. We must face the question and deal with it in the large. The percentage of risk does not appear to be very high, especially if suitable care be taken. On the other hand, the condition for which we are undertaking operation is an extremely serious one, and justifies us, up to a certain point at least, in taking some chances.

(iv.) There is a hesitation amongst surgeons in undertaking any new form of procedure. This attitude is not merely comprehensible, but within limits it is justifiable. Time and a favourable experience alone will serve to break it down.

CHAPTER IX

SECONDARY GLAUCOMA

The study of secondary glaucoma is of importance to the general practitioner for three reasons.—

1. It throws a valuable light on the ætiology of the primary form of the disease.

2. It shows the need for early and careful diagnosis.

3. It emphasizes the fact that it is difficult to treat cases of this kind, for the obvious reason that we must not only strike at the rise in intra-ocular pressure, but must also, and in the first instance, direct our attention to the causative condition, which is not seldom very intractable.

Ætiology.—The causes of secondary glaucoma may be divided into two groups—viz., (*a*) those which act by bringing about a closure of the angle of the anterior chamber, and thus shutting off the stream of fluid from the excretory outlets in this neighbourhood; and (*b*) those which owe their influence to a change in the constitution of the intra-ocular fluid. Such change may be either due to an alteration in the composition of the fluid (*e.g.*, from the presence of albuminous matter in solution therein), or to the presence of epithelial or other débris therein. Both these changes tend to block the excretory channels mechanically, and so to impede the outflow of aqueous fluid through them.

Secondary glaucoma may be brought about by any of the following conditions.—

Fistula of the Cornea.—Whilst the fistula is open, the anterior chamber lies empty for a longer or shorter period, with the iris base in contact with the cornea; the formation of exudate, as a result of inflammatory action, may agglutinate the two apposed surfaces, and so cause obliteration of the angle of the chamber. When the fistula heals, and the fluid can no longer escape through it, the normal channels may be found to be occluded, with the result that the intra-ocular pressure rises. Later, staphyloma of the cornea may follow.

Anterior Synechia, following a fistula or a penetrating injury, may cause such displacement forward of the iris base as to effect an obliteration of the angle of the chamber. Such a closure is usually partial in the early stage, and may become complete later.

Posterior Ring Synechia leads to *l'iris bombé*, a condition in which the iris is pushed forward by the fluid dammed up behind it. The result is an obliteration of the angle of the chamber, which adds to the difficulty already caused by the want of a free circulation through the pupil.

Anterior Dislocation of the Lens.—Here the iris is found wrapped round the posterior surface of the lens, and also in close contact with the cornea peripherally, thus sealing the angle all round.

Lateral Dislocation of the Lens acts much like anterior synechia, only here the force that closes the angle is a push from behind instead of a pull from in front. It also resembles the latter condition in that the occlusion is partial in the first instance at least.

Injury to the Lens.—As a result of accidental trauma, the capsule of a lens may be ruptured or pierced. In either case the lens fibres swell up, with the result that the lenticular mass increases in bulk, and so comes to press upon the angle of the chamber and occlude it.

In addition, the lens masses tend to pass into solution, and so to change the constitution of the aqueous, rendering it less able to pass through the meshes of the pectinate ligament. We have thus a twofold tendency to secondary glaucoma.

Operations.—As a result of accidental injury during operations, the same sequence of events, as has above been outlined, may take place. Moreover, in the discission of soft cataracts the capsule is deliberately cut over a limited area, with a view of letting in the aqueous fluid; the swelling produced may sometimes outrun our expectations or wishes, and then secondary glaucoma follows.

After Cataract Extraction a dangerous form of secondary glaucoma is sometimes met with. It may be due to any of the following causes:—(1) impaction of a tag of capsule in the section; (2) closure of the pupil by exudate; (3) the formation of a membrane behind the pupil, which becomes impermeable to fluid, and so stops the passage of the aqueous forwards; (4) the presence of a slow irido-cyclitis, with all that this connotes; (5) the passage of vitreous fluid forward into the angle of the chamber.

Intra-ocular Tumours.—These set up a low form of uveitis, as a consequence of the liberation of toxic substances by the growing organism. In this way the angle may become sealed.

Cyclitis acts in two ways:—(1) by sealing up the angle of the chamber with exudate, and (2) by altering the contents of the aqueous. The chemical constitution of the fluid may be changed, rendering it less liable for filtration; or cellular contents may be added to it, which tend mechanically to block up the meshes of the pectinate ligament.

Intra-ocular Hæmorrhage adds suddenly to the volume of the intra-ocular contents; it also makes the vitreous

more albuminous, and so promotes an osmotic flow into it from without.

The Diagnosis of Secondary Glaucoma.

In each individual case the signs and symptoms of the primary disease must be carefully noted and their significance weighed. The conditions which may give rise to this form of glaucoma have already been sufficiently indicated in the previous paragraphs.

The Treatment of Secondary Glaucoma.

In all cases in which the primary disease is still active, it should be most energetically attacked, regard being had not merely to symptomatic treatment, but above all to the root-cause of the condition. To take one instance, the treatment of glaucoma secondary to cyclitis is incomplete if due attention be not paid to the possibility that pyorrhœa, latent gonorrhœa, syphilis, or some other form of auto-intoxication, may be at the root of the matter.

When the iris base is drawn forward, and the condition is an early one, iridectomy is indicated. This is especially the case in anterior synechia.

Pressure by a dislocated lens or by masses of lens matter, following any form of trauma, demands the free evacuation of the offending material.

Glaucoma secondary to the swelling of a maturing cataract is best met by a prompt iridectomy. That due to the formation of an impervious after-cataract may sometimes be relieved by free discission of the obstructing membrane. Glaucoma following cataract extraction, in the absence of any such membrane, often yields promptly and permanently to trephining. Where, however, it is due to the vitreous passing freely through the pupil into

the anterior chamber, and there proving too viscid to allow of the natural escape of the fluid, it is doubtful if anything can be done.

The presence of an intra-ocular tumour indicates the need for an excision of the eyeball.

In hæmorrhagic cases medicinal and general treatment are probably the safest, though under desperate circumstances it may be permissible to trephine.

The treatment of iritis and cyclitis is on ordinary lines; paracentesis, however, should always be kept in reserve.

There are few more difficult things to decide than the best method of *immediate* treatment of a case of acute or subacute congestive glaucoma. The use of eserine, which will open the angle, will at the same time increase the congestion present. Atropine will reduce the congestion, but will increase the difficulty opposed to the outflow of the intra-ocular fluid. Operation in any form is dangerous, though it may have to be undertaken. There are cases in which the bold use of atropine or of eserine, selected according to the exigencies of the individual case, may prove invaluable, and there are times when operation must be undertaken and the fate of the eye risked on the result of a single throw. The practitioner will be well advised if, under such circumstances, he places the responsibility on an expert, if possible. In the meantime the patient should be kept in bed, the forehead and temple should be freely leeched, and vigorous fomentations applied. Free purgation should be established, but not pushed after the first twenty-four hours. Morphia should be used, if not otherwise contra-indicated, to relieve pain and to obtain sleep, and the patient should be kept quiet, in a shaded light. Needless to say, all the individual indications previously dealt with should be carefully attended to. The surgeon must

always remember that the aim of his treatment is not merely to bring the patient through a painful period into a condition of physical quiescence, but also, and above everything else, to *save as much vision as possible.* If he fails in this last respect, he has practically failed altogether.

CHAPTER X

CONGENITAL AND JUVENILE GLAUCOMA

CONGENITAL GLAUCOMA

By this we understand glaucoma which is congenital in origin. A large percentage of such cases are also congenital in their manifestation of the condition, though the earliest signs of many such are undoubtedly overlooked both by medical men and by parents. At the other pole of life—say from the age of forty onward—are found the cases of senile glaucoma. Intermediate between the two, too late to be classed as congenital, too early for the senile group, are a number which, for convenience' sake, we call "juvenile." Speaking generally, congenital glaucoma is due to defects in the development of the excretory passages, whilst senile glaucoma is to be attributed to degenerative and other changes which accompany the advance of age. It is probable that both elements play a part in the juvenile cases, and the preponderance of one or the other influence determines whether the case is an early or a late juvenile one, and so whether it is in danger of being confused with those of the congenital or senile classes. The terms "buphthalmos" and "hydrophthalmos" are synonyms of congenital glaucoma.

Signs and Symptoms of Buphthalmos.

The condition is usually noticed at birth or before the third year, and is bilateral in the great majority of cases. Boys are more frequently affected than girls.

The eye enlarges slowly but steadily, and may attain enormous proportions. The *whole globe* is uniformly distended. Proptosis, lagophthalmos, and limitation of ocular movement follow. The great enlargement of the *cornea* is a striking and obvious feature. That membrane may retain its lustre, or it may become scarred in various ways.

The *anterior chamber* is greatly deepened. The *sclera* is evenly distended, and the uveal pigment shows through the thin coat, making the white of the eye appear of a bluish colour. The *iris* is tremulous; the pupil is round, sluggish, and slightly dilated. The *lens* is normal in size or smaller than natural, and is tremulous owing to the overstretching of its suspensory ligament. The *vitreous* may be clear or more or less cloudy. The *choroid* is overstretched and atrophic. The *retina* is overstretched, and detachment may take place. The *optic disc* is cupped. The *tension* of the eye is raised, sometimes very markedly so; owing to the alteration in the curve of the cornea, the ordinary tonometer cannot be used, and we must rely on a finger examination. The *visual acuity* is often much impaired owing to changes in the media and in the optic nerve and retina. A moderate myopia is usually present, often associated with astigmatism, but its correction by glasses is difficult. *Nystagmus* is common. The children are usually backward and diffident, and their general *health* is often below *par*.

Clinical Course and Complications.

The great majority of cases go steadily downhill to loss of vision, either through retinal degeneration or opacification of the media, or both. A few cases stop spontaneously; yet others, and these fewer still, retain throughout comparatively normal functions. It has been

suggested that the last class represent a gigantism of the eye, for which the name "megalocornea" has been proposed. This is a disputed question. The buphthalmic eye is very liable to injury. In a few cases buphthalmos is associated with an obscure condition termed "neurofibromatosis."

Differential Diagnosis of Buphthalmos.

Buphthalmos must be distinguished from megalocornea, juvenile glaucoma, keratoconus, keratectasia, staphyloma, and exophthalmos. The first two conditions have already been dealt with. In the remaining four the diameter of the corneal base is not increased, whilst in congenital glaucoma it is above the normal and continues to enlarge.

The Pathological Anatomy and Ætiology of Buphthalmos.

The leading points are as follows.—(1) The *cornea* is very liable to damage, especially on its posterior surface, due to overstretching and consequent tearing of the membrane of Descemet. This gives rise to characteristic scars at the back of the cornea. (2) *The anterior chamber* is very deep, and its angle appears to be widely open. This appearance is often deceptive, as has been proved by anatomical examination, for the angle is frequently found to be closed by bands in this neighbourhood. These represent a fœtal condition of development. The *canal of Schlemm* is often absent or insufficiently developed. It is this deficiency of the normal passages for the excretion of intra-ocular fluid which is the paramount factor in the ætiology of buphthalmos. The influence of *heredity* is well marked, and is probably a question of the inheritance of the above-mentioned defective develop-

ment. The next point of importance is the *distensibility* of the young eye, which accounts for and permits the great enlargement characteristic of the disease, and which is not present in older globes.

Buphthalmos not seldom follows an injury or inflammation of the eye. In most of these cases the condition is probably quite distinct from that of congenital glaucoma; they really are instances of secondary glaucoma occurring in young and therefore distensible eyes.

JUVENILE GLAUCOMA

This group comprises a great variety of forms, some of which are really congenital in origin (though their onset may be delayed or may at first escape observation), whilst others closely resemble the different forms of senile glaucoma. Every variety of senile glaucoma may be imitated, from the simple to the most acute. A very interesting feature is the common association of juvenile glaucoma with myopia. Another is the tendency for the early stage of the disease to be prolonged owing to the fact that the yielding of the young globes staves off, for a time at least, the worst consequences of the rise in intra-ocular pressure.

It cannot be too strongly insisted on, that there is no essential difference between the various forms of glaucoma. The best evidence of this is found in the following facts. When glaucoma affects several successive generations, it tends to be of the senile type in the first generation, and more and more of the juvenile type in the later ones. Again, in a single generation of one family instances may occur of juvenile glaucoma in some members, and of buphthalmos in others. Lastly, a single individual may have buphthalmos in one eye, and juvenile glaucoma in the other.

The Treatment of Congenital and Juvenile Glaucoma.

The treatment of these conditions by medicine has been very unsatisfactory. Inasmuch as some few of them either never lead to serious symptoms, or else tend to come to a full-stop by themselves, it is well to watch each patient closely, and only to decide on active interference when the case is steadily going downhill. The reliance to be placed on meiotic and similar treatment is " expectant " only of disaster. If operation is decided on, it should be undertaken at the earliest possible moment. Iridectomy has been practically abandoned as unduly dangerous in these conditions.

Many forms of operation have been tried, with varying success. Sclerectomy has proved more satisfactory than sclerotomy or anything else, and trephining appears to have yielded the best results of any procedure. It would, none the less, be idle to conceal the fact that, do what we may, the prognosis of a case of buphthalmos is not a very happy one. On the other hand, the dismal certainties that attend inaction make it imperative that we should not hesitate to shoulder our responsibilities towards the patients and their relatives. The author's personal experience has shown him that a certain number of cases will yield results which, if not brilliant, are at least highly gratifying, inasmuch as they enable a child to see and to play a useful part in life. It is certainly worth operating on ten cases to get five such results, and that is, we think, somewhere near the probabilities of the matter.

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